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CLINICAL PRACTICE
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Multiorgan evaluation of perfusion and congestion using ultrasound in patients with shock

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There is increasing evidence on the role of ultrasound in the evaluation of multiorgan hypoperfusion and congestion in patients with cardiocirculatory shock both to identify the underlying pathophysiological mechanism and to drive and monitor the treatment. The cardiac and lung ultrasound is included as an integrated multiparametric approach to the very early phase of patients with haemodynamic instability/cardiogenic shock. Splanchnic ultrasound has been mainly applied in heart failure and predominant circulatory failure. Although poorly validated in the critically ill, many ultrasound parameters have a strong physiological background to support their use in the acute setting those that apply either for heart/lung and for splanchnic organ evaluation. This review summarizes the ultrasonographic parameters that have shown evidence in literature in the diagnostic/therapeutic pathway to define the congestion/perfusion profile of the organs that are involved in the pathophysiological cascade of cardiocirculatory shock.

Keywords Ultrasound • Congestion and perfusion • Shock • Echocardiography • Splanchnic Doppler

Introduction

Shock is the ultimate clinical manifestation of circulatory failure. It is a term which encompasses a multitude of physiological perturbations, all of which ultimately result in inadequate cellular oxygen usage. Shock in all its forms is ubiquitous in the critical care arena and is present in approximately one-third of patients admitted to the intensive care unit (ICU).[1] The consequence of shock, impaired end-organ circulation, may occur because of either inadequate flow to the organ (perfusion), because of impaired flow from the organ (congestion), or more often, because of an amalgamation of both phenomena. While reduced global oxygen delivery (DO_2) is often attributed to diminished tissue perfusion, congestion likewise reduces the arteriovenous gradient across vital organs driven by increased interstitial pressure rather than low arterial pressure.

While protocolized approaches to patient management advocated in the last two decades have certainly helped with disease-specific awareness and screening, there are inherent shortcomings to a one-size-fits-all strategy. Often, a personalized approach in response to present bedside physiology is required for management. Comprehensive patient assessment should encompass several modalities; however, certainly, the most powerful and versatile of these tools is point-of-care ultrasound (POCUS).

The role of POCUS in the acutely ill patient is to identify present and relevant physiology, otherwise known as 'phenotyping shock'. Once the type of shock has been identified, targeted management can begin. Astute diagnostics are a poor substitute for source control. Rather, identifying the source of the patient's shock and endeavouring to intervene is paramount to the management and resolution of shock. Herein, we review organ-specific POCUS assessments of both perfusion and congestion while discussing the physiological rationale and the available evidence for the use of these in clinical management. A summary of commonly employed perfusion and congestion morphologies is seen in *Figure 1*. Practical approaches to perform each technique are found in the supplement.

Perfusion deficit

The first step in the evaluation of a patient's circulatory performance starts with both quantitative and qualitative assessment of the delivery of blood to target organs, otherwise known as perfusion. A perfusion

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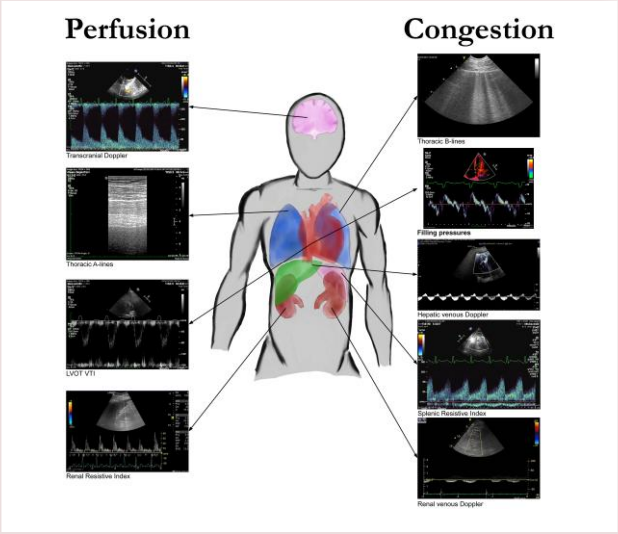

Figure 1 The multiorgan approach to evaluate congestion and perfusion with relevant parameters discussed in the text.

mismatch may be present by a primary alteration of cardiac function (cardiogenic shock) or may be related to predominant circulatory dysfunction. Therefore, the evaluation of congestion/hypoperfusion should encompass all the organs potentially involved as each modality of ultrasound confers actionable data points in the sonographic assessment of the heart, lung, abdominal viscera, and brain.

Evaluation of perfusion

Heart

Echocardiography is the cornerstone of bedside ultrasound assessment of cardiac function and global perfusion as it both defines relevant haemodynamic parameters and identifies a potential aetiology for patients with shock. Significant effort has been set forth to identify training requirements and echocardiography standards.[2] The clinician's challenge is to contextualize echocardiographic findings to the patient's clinical picture and adjust ongoing supports accordingly (inotropes/vasopressors, positive pressure ventilation, mechanical circulatory support). Patients with myocardial dysfunction present a unique challenge to the intensivist. Both LV and RV dysfunction may represent the primary cause leading to shock or either represent an epiphenomena of a different pathological process including/leading to cardiocirculatory failure.

Haemodynamic assessments have historically equated cardiac function to LV function. Over the past decade, the prevalence and the consequences of RV dysfunction in disease states such as sepsis and acute respiratory distress syndrome have become more apparent.[3,4] A nuanced approach to haemodynamics incorporates both LV and RV function to provide an accurate haemodynamic profile.[5]

The left ventricle (LV) acts four principal movements for each contraction: circumferential, radial, longitudinal, and oppositional torsion between apex and base. The synergy of those results in ejecting flow from the LV to the systemic circulation. Echocardiography quantifies the performance of each of the cardinal directions of LV function; however, assessment of the longitudinal function of the LV is the most tangible by bedside echocardiographers. Left ventricular ejection fraction (LVEF) and stroke volume (SV) are the most commonly used metrics in point-of-care echocardiography centred on the assessment of forward flow. Left ventricular ejection fraction has been used for over 60 years for clinical classification, decision-making, and prognostication.[6] Despite LVEF's hallowed position as the most commonly used descriptor of heart failure, in practice, LVEF relies on several assumptions which diminishes its utility in the critical care setting (*Table 1*).

Left ventricular ejection fraction is a volume-derived measurement predicated on often-flawed geometric assumptions such as synchronous, uniform systolic function of LV walls.[9] Such assumptions are violated in the setting of regional ischaemia, myocardial infarction, bundle branch blocks, and arrhythmia resulting in an unreliable estimation of LV function.[10] Additionally, LVEF acutely varies in response to haemodynamics such as heart rate, preload, afterload, and inotropy, all of which are commonly adjusted in the critical care arena. The LVEF represents either a summation of all three directional components (Simpson's rule) or both circumferential and radial components (Teichholz formula) but does not explicitly address longitudinal function. The extremes of LVEF have been associated with mortality in patients with septic shock.[11]

Left ventricular longitudinal fibres reduce the dimension from the LV base to the LV apex during systole, contributing up to 30% of LV function.[10] One metric of LV longitudinal function is mitral annular plane systolic excursion (MAPSE) assessed either with m-mode and tissue Doppler imaging (TDI). Mitral annular plane systolic excursion leverages a simple technique with reasonable correlations to other metrics of LV function such as LVEF when measured at the level of all ventricular basal walls.[12]

Mitral annular plane systolic excursion is particularly sensitive to early coronary hypoperfusion as it represents contraction of the subendocardial fibres of the LV, which are the most susceptible to coronary ischaemia.[13] This may result in a reduction of longitudinal excursion and a prolongation of systolic contraction, in turn, leading to concomitant diastolic abnormalities.[14] Diastolic impairment occurs early in the myocardial ischaemic cascade. Mitral annular plane systolic excursion likely correlates with mortality for patients with shock.[12] Despite the understood performance of MAPSE, clinicians should maintain caution to not draw conclusions on the global function of the LV based on a local measurement.

The systolic peak velocity (S′) is measured by TDI at the same location on the lateral wall as MAPSE. The systolic peak velocity measures the mobility of the lateral wall by speed rather than by measuring its displacement. The S′ correlates with *dP/dT*, and it is relatively independent from loading conditions.[15] A reduction in S′ velocity is detectable within 15 s of the onset of ischaemia. The presence of S′ post-systolic shortening was associated with adverse remodelling and hospitalization for patients with ischaemic cardiomyopathy.[16–18]

The direct measurement of stroke volume circumvents several shortcomings encountered by using LVEF as a metric for perfusion. The calculation of the area under the velocity–time curve produced by placing the sample volume of the pulsed wave Doppler in either the right ventricular outflow tract (RVOT) or the left ventricular outflow tract (LVOT) yields the velocity–time integral (VTI), a value proportional to stroke volume (see Supplementary material online, *Figures S1* and *S2*). The VTI from either the RVOT or LVOT is one of the most reliable and reproducible echocardiographic parameters used to estimate stroke volume.[19–21] As such, at similar heart rates, serial measurements of LVOT VTI correspond to changes in stroke volume and can be used to monitor responses to interventions. Changes in LVOT VTI have high predictive values in fluid responsiveness in septic shock and mechanically ventilated patients as well as value of predicting successful weaning from venoarterial extracorporeal membrane oxygenation (V-A ECMO).[22,23]

There are additional echocardiographic parameters that have demonstrated high accuracy in describing ventricular performance and/or haemodynamic profiles as related to outcomes in the shock. Such parameters such as LV total isovolumic time (LV t-IVT-Supplementary material online, *Figure S3*), stroke work index, and

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Table 1 Ultrasound parameters to assess perfusion

VARIABLES	*Abnormal cut -off values*	*Transducers*	*Indication*	*Advantages*	*Limitation*	*Interobserver agreement/ variability*
LEFT VENTRICULAR EJECTION FRACTION (LVEF)	HFrEF < 40%; HFmrEF 41–49%; HFpEF > 50%	Phased array (2–12 MHz)	Left ventricular systolic function prognostic implication in CHF	Widely used; volumetric evaluation of radial/ circumferential shortening; visual assessment; regional wall motion abnormalities	Heart rate, preload and afterload; loading conditions; dys-/asynchrony; delayed contraction Severe mitral regurgitation and aortic stenosis Severe hypertrophy; inconsistencies in the tracing of the LV cavity Requires geometric assumptions	Simpson's: ICC 0.7 and Bland–Altman from ± 7% to ± 25% Visual: ICC 0.87
MAPSE	< 10 mm in each annular region (anterior, posterior, lateral, inferior)	Phased array (2–12 MHz)	Global change in size of the LV cavity in the long-axis direction; prognostic implication in AF, post-MI, HF	Highly reproducible also with sub-optimal windows; predictive of symptoms in AS	LBBB, paradox septal motion and aortic valve replacement affect septal MAPSE; mitral calcification/ prosthesis. Large pericardial effusion (mobile apex), severe hypertrophy	CoV 9.3 6 ± 6.9%.
TAPSE	< 17 mm	Phased array (2–12 MHz)	Right ventricular longitudinal function	Intrinsic longitudinal contractility; validated in wide range of patients categories; highly reproducible; not affected by dropout or trabeculations	Load dependent, does not reflect RV 3D contraction	ICC 0.83 (0.67–0.91)
TOTAL ISOVOLUMIC TIME (T-IVT)	≥ 14 s/ minutes[7]	Phased array (2–12 MHz)	Electro-mechanical efficiency; global mechanical dyssynchrony	Reproducible; early; systolic and diastolic period assessment; independent by morphological and functional (valvular) alterations; more sensible than MPI	Not validated in arrhythmias nor in severe hypertrophy	CoV 4.2–6.4%.[8]
VENTRICULO-ARTERIAL COUPLING (VAC)	>1.36[84]	Phased array (2–12 MHz)	Interaction between the LV and aorta	Feasible, multiparametric, integration between cardiac and vascular component	Imaging methods relies on LVEF, validity unknown in arrhythmias and severe hypertrophy	Not specified; however linked to LVEF and time intervals measurements

Continued

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Table 1 *Continued*

VARIABLES	*Abnormal cut -off values*	*Transducers*	*Indication*	*Advantages*	*Limitation*	*Interobserver agreement/ variability*
LEFT VENTRICULAR STROKE WORK INDEX (LVSWI)	≥50 g × min/ m2	Phased array (2–12 MHz)	Evaluation of ventricular stroke work index	Incorporates both LV systolic and diastolic function to quantify the ability of the LV to generate forward flow and pressure; validated in CS patients	Relies on LVEDP estimated with echo; not validated in therapy monitoring	Not specified
CARDIAC POWER INDEX (CPI)	0.6–0.7 W/m^2	Phased array (2–12 MHz)	Evaluation of cardiac pump ability	Reflects the energy applied to generate stroke volume; validated in CS patients	In CS, cohort described RAP was not taken into account	Not specified
RENAL DOPPLER RESISTIVE INDEX (RDRI)	0.7	Convex probe (3–6 MHz)	Kidney arterial perfusion	Highly reproducible; early detection of haemodynamic abnormality independent of angle and vessel CSA	Reduced tissue compliance; interstitial fibrosis; reduced vascular compliance; sampling inaccuracy; atrial fibrillation	ICC 0.96
SPLENIC DOPPLER RESISTIVE INDEX (SDRI)	>0.55	Convex probe (3–6 MHz)	Splenic perfusion	Backward pressure of splanchnic compartment; independent of Doppler angle and vessel CSA	Chronic liver disease, altered parenchymal compliance	Not enough data in critical care setting
PORTAL FLOW PULSATILE INDEX (PFPI)	>50%	Convex probe (3–6 MHz)	Liver perfusion and systemic venous congestion	Inter-play between systemic perfusion, RV and pulmonary function, and venous hypertension	Advanced liver primary disease; high intrathoracic pressure, respiratory distress, portal shunts	ICC 0.82

Heart failure with reduced ejection fraction, HFrEF; HFmrEF, heart failure with midrange ejection fraction; HFpEF, heart failure with preserved ejection fraction; CHF, chronic heart failure; post-MI, post–myocardial infarction; ICC, intra-class correlation coefficient; AF, atrial fibrillation; AS, aortic stenosis; LBBB, Left bundle branch block; CoV, coefficient of variation; LVEDP, left ventricular end-diastolic pressure; CSA, cross-sectional area.

cardiac power output describe LV performance; however, prospective large-scale validation in patients with shock is lacking.[8,24,25]

Right ventricular echocardiography presents unique challenges owing, in part, due to its complex geometry and its proximity to the lungs. As such, commonplace LV measurements such as ejection fraction and fractional area change which require clear endocardial border definition are frequently unobtainable for the right ventricle (RV) , especially for patients who are mechanically ventilated. The most commonly reported RV metric in point-of-care echocardiography is RV size relative to LV size (the RV/LV ratio), an indicator of RV overload. The qualitative estimation of RV systolic function although frequently reported in practice lacks sensitivity and poor interobserver agreement.[7] Quantitative RV metrics are analogous to the aforementioned LV metrics. Tricuspid annular systolic plane excursion (TAPSE) and TDI RV S′ at tricuspid annulus reflect RV longitudinal fibre shortening and reflects RV contractility. Both TAPSE and RV S′ are predictors of MACE and mortality in a wide range of patient populations.[26]

Using the modified Bernoulli equations, it is possible to estimate cardiac chamber and pulmonary artery (PA) pressures. While RV systolic, PA systolic, and PA diastolic pressures are commonly reported in complete echocardiograms, such values are seldom helpful to determine the source of shock. Rather, transvalvular gradients may indicate the presence of elevated filling pressures which highlight intolerance to fluid resuscitation. Estimation of right atrial pressure (RAP) itself often does not confer actionable clinical information.

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While there are several additional echocardiographic metrics which reveal unique aspects of myocardial performance, their interpretations are often limited by patient factors common to the intensive care environment such as tachydysrhythmias and mechanical ventilation. Future directions in assessing myocardial performance in the critical care arena include speckle tracking and transoesophageal and intracardiac echocardiography.

One can assume hypoperfusion of end organs is generally homogeneous; however, when clinical scenarios indicate poor end-organ performance, targeted assessments of end-organ perfusion may provide additional insight. Once a patient's shock has been appropriately phenotyped, clinicians should titrate the ongoing medical therapy to achieve clinical goals.

Lung

Ensuring adequate transpulmonary blood flow should be an element of routine sonographic evaluation for patients with shock. Transpulmonary blood flow requires a favourable pressure gradient from the RVOT to the left atrium. Analysis of the RVOT pulsed wave Doppler morphology and its acceleration velocity indicate the presence and/or severity of pulmonary hypertension.[27] Consequently, interrogation of the RVOT with pulsed wave Doppler allows clinicians to not only estimate RV stroke volume, but also gain insight into resistance of the pulmonary vasculature and potential response to interventions.

Left atrial (LA) pressure can be inferred by several different echocardiographic modalities including transmitral Doppler, diastolic evaluation with TDI, pulmonary venous Doppler, and thoracic ultrasound. Pulmonary vein Dopplers provide insight into left-sided chamber performance and can be obtained in ~90% of non-critically ill adults.[28,29] Additionally, there is significant homology between the interpretation of pulmonary venous and hepatic venous Doppler waveforms. On thoracic ultrasound, A-lines may represent dry parenchymal septae and have been correlated to low pulmonary artery occlusion pressures.[30]

Abdomen

In shock states, perfusion to the heart and brain is often maintained at the expense of the splanchnic circulation resulting in visceral hypoperfusion. Perfusion to abdominal organs can be interrogated using typical modalities of ultrasound including 2D brightness, colour Doppler, and pulsed wave Doppler. The Doppler resistive index (DRI) evaluates regional splanchnic haemodynamics and may provide early detection of abnormalities related to organ dysfunction before biochemical and macro-haemodynamic are evident, thus indicating the adequacy of systemic circulation to support splanchnic perfusion (see Supplementary material online, *Figure S4*).[31,32] In the setting of splanchnic vasoconstriction, there is an increased tissue resistance to flow resulting in a slowing the diastolic velocity, thus increasing the DRI. The DRI can be used for several splanchnic viscera and is easily calculated [(peak systolic velocity–trough diastolic velocity)/peak systolic velocity].[33]

For patients experiencing hypovolaemia, renal blood flow can be reduced up to six times from baseline. The renal resistive index (RRI) allows for the assessment of renal perfusion and is influenced both by intra-renal conditions and by haemodynamic conditions. The RRI leverages high feasibility, predictive value of acute kidney injury development in critically ill, and low intra- and interobserver variability.[34,35]

In the settings of hypoxia or in conditions that predispose to hypercapnia, a significant renal blood flow reduction is often seen. An inverse correlation exists between SvO_2 and RRI as expression of renal high sensitivity to oxygen supply/demand mismatch leading to arterial vasoconstriction. A RRI $\geq$ 0.70 was 100% specific in predictions of an SvO_2 < 60%.[33] Predictably, the RRI identifies hypoperfusion related to reduced C_AO_2 in mechanically ventilated patients prior to haemodynamic and ventilatory parameter variations.[36,37] Elevated values of RRI may demonstrate increased metabolic needs in patients with euvolaemic anaemia as well as for patients with relative hypovolaemia before developing overt signs of shock.[38] For patients with septic shock, RRI has been used to establish optimal renal perfusion pressure by way of vasoactive infusion titration.[39] Specifically, an RRI > 0.77 predicted sepsis-induced AKI and an RRI > 0.80 predicted a persistent AKI.[40,41]

Novel renal arterial measurements including pre-ejection period (PEP), ejection time (ET), and PEP/ET may indicate inadequate cardiac output to meet metabolic demand of the kidney, thus potentially able to predict adverse cardiac and renal outcomes.[42–44]

Central nervous system

Transcranial Doppler (TCD) is a routine technique to assess blood flow to the brain. There are four standard TCD views; however, the transtemporal view is the most commonly used in ICU. Transcranial Dopplers have historically been employed for patients who have sustained either haemorrhagic or ischaemic insults which impact central nervous system (CNS) haemodynamics.[45]

There is a dearth of literature regarding the application and manipulation of TCDs in patients with shock. The majority of literature regarding sonographic examination of CNS blood flow is for patients who require mechanical circulatory support. For patients with cardiogenic shock (CS) supported by various configurations of V-A ECMO, TCDs have been used to examine cerebral blood flow as a surrogate for native cardiac output.[46] Additionally, for patients with CS supported by V-A ECMO with an intra-aortic balloon pump as an LV vent, TCDs have been used to demonstrate augmented blood flow during support.[47] For patients on V-A ECMO with pulsatile flow, TCDs have been used to indicate brain death.[48]

In septic shock, cerebral perfusion is altered by way of activation of endothelial cells by pro-inflammatory cytokines and endotoxins promoting an early vaso-constrictory response followed by a late vasodilatation with increased cerebrovascular flow.[49] As such, unique TCD patterns can be detected depending on different sepsis stages with a prevalent increase in mean velocities and pulsatility index in the early phases followed by a decrease in the late stages along with the impairment of cerebral autoregulation. Thus, TCD has been suggested as a valuable tool for the assessment of cerebral perfusion in septic shock.[50] A relationship between congestion and CBF has not been described systematically although some insight based on local experience has been described.[51]

Venous congestion

Congestion is defined by a state of venous excess where retrograde flow of blood away from the heart is observed, causing congestion at the organs, leading to a decrease in perfusion pressure across the capillary bed. To fully understand the determinants of venous congestion, one must first understand the physiology of venous return. The physiological determinants of venous return are the components that make up the mean systemic filling pressure (MSFP) and the RAP and the resistance of the venous system (MSFP-RA/R).

As the venous system is primarily a capacitance system, resistance rarely plays a major role in determining venous return. Thus, the major determinants of venous return are the MSFP and the RAP (MSFP-RA). Mean systemic filling pressure is created by the volume of blood in the venous system at any given time and the vascular tone of the vessels. If one were to graph MSFP and RAP on the same pressure time, the difference between the two lines on the *y*-axis would illustrate the direction of blood flow to and from the heart throughout the cardiac cycle.

During right atrial (RA) systole, RAP increases above MSFP and blood flows retrograde away from the heart into the venous system. Once the right atrium (RA) relaxes, the pressure in the RA decreases and blood flow starts to flow back towards the heart. Right ventricular systole causes a rapid decrease in RAP as the RV contraction pulls the tricuspid valve apically. This leads to an abrupt drop in RAP, increasing the

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flow of blood from the venous system towards the heart. As blood fills the RA, pressure rises and flow towards the heart decreases. Right ventricular diastole begins as the tricuspid valve opens, and blood flows from the RA to the RV. This leads to a decrease in RAP once again, increasing blood flow back to the heart. At this point, the RA again contracts, starting another cardiac cycle. This represents normal venous return during a standard cardiac cycle.

As a patient develops an increase in RAP, the relationship between the MSFP and RAP shifts. Retrograde flow during RA systole increases. Conversely, the anterograde flow that normally occurs during RV systole decreases to a level less than what occurs in RV diastole. This is what is known as S-to-D inversion. In severe cases, RAP increases to levels where it spends the majority of the cardiac cycle above the MSFP. In these cases, retrograde flow occurs throughout RV systole, leaving RV diastole the only period where venous return occurs.

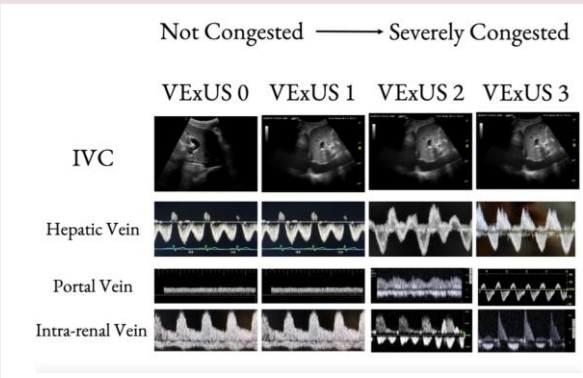

Figure 2 Venous excess ultrasound protocol.

Evaluation of congestion

Heart

The sine qua non and indication of worsening heart failure is LA hypertension and concomitant congestion.[52] Left atrial hypertension results in pressure transmitted to nearby vasculature, namely, the pulmonary veins and pulmonary capillaries resulting in pulmonary oedema. Left atrial hypertension of chronicity results in pulmonary hypertension which results in right-sided chamber distension.

While it is understood that with both systolic and diastolic dysfunction that transmitral velocities and tissue Doppler values become abnormal, established normal values of diastology are not validated in the critically ill. Additionally, all echocardiographic modalities to discern LA pressure provide only a range of values rather than a value itself. For patients with septic shock, however, an average $E/e' \geq 14$ and an $e' \leq 8$ reliably correlated with a pulmonary capillary wedge pressure (PCWP) > 15 mmHg and was a predictor of in-hospital mortality.[53] In mechanically ventilated patients, E/e' and colour M-mode propagation velocity performed most optimally to identify a pulmonary artery wedge pressure > 18 mmHg.[54] If diastolic function is indeterminate, many clinicians use LA size as a 'tiebreaker' to clarify the presence of elevated filling pressures. Left atrial enlargement suggests a chronically elevated LA pressure; however, a normal LA size should not exclude LA hypertension especially in acute illness.[55]

The most commonly employed metric of venous congestion is RAP or central venous pressure (CVP). Elevated RAP is usually estimated by an evaluation of inferior vena cava (IVC) dimension (>2.1 cm) and its variations over the respiratory cycle in spontaneous and mechanically ventilated patients (respectively, > 50% and >20% to predict fluid responsiveness). Analogously, the internal jugular vein (IJV) was studied in patients with septic shock and found that distensibility more than 18% prior to volume challenge had an 80% sensitivity and 85% specificity to predict fluid responsiveness.[56] Several common clinical conditions (i.e. respiratory distress, high intrathoracic pressures, severe tricuspid regurgitation) limit the utility of static metrics such as an isolated RAP value.[57]

Lung

Lung ultrasound (LUS) consists of the evaluation of the parenchyma and pleura. Lung ultrasound permits us a view of venous congestion's effects beyond the vascular space. It is important to note this is representative of congestion of the pulmonary venous system rather than the systemic venous system. First introduced by Lichtenstein in patients with acute respiratory failure admitted in general intensive care and since the last decade, there is an increasing body of evidence demonstrating its value as both a diagnostic and prognostic tool. The interpretation of LUS is based on two major pathological findings: solid pictures (consolidations) and artifact (B-lines) in comparison with normal lung findings (A-lines).

B-lines have been found to correlate well with the other markers of extravascular lung water, including pulmonary wedge pressure and natriuretic peptides. Lung ultrasound has been demonstrated to be a highly accurate diagnostic tool in patients presenting in respiratory distress differentiating between dyspnoea from cardiac or non-cardiac origin.[58] More importantly for the current discussion, LUS is a reliable dynamic marker of parenchymal congestion during decongestion therapy, stratifying and predicting prognosis in patients with heart failure and acute myocardial infarction CS.[59,60] This allows clinicians to identify signs of venous congestion in real time during an active resuscitation, potentially limiting the harms of further fluid resuscitation. The integration of echocardiography and LUS carries higher accuracy in the diagnosis and the origin of respiratory failure in the critically ill.[58]

Abdominal viscera

Hepatic vein Doppler

Hepatic venous flow can be interrogated using pulsed wave Doppler. Its normal flow curves mirror the theoretical venous return curves discussed in the previous section. The normal waveform is triphasic with a retrograde A-wave (representing blood flowing away from the heart), an antegrade S-wave, and an antegrade D-wave (see *Supplementary material S5*).

With an increase in RAP, a smaller S-wave is observed as equilibration between MSFP and RAP occurs earlier. Due to this early equilibration, more RA filling occurs after the tricuspid valve opens, leading to a relatively larger D-wave. This is what is known as S-to-D reversal. As venous congestion worsens, systolic flow becomes retrograde, and the S-wave will move above the baseline becoming continuous with the A-wave leading to a biphasic pattern.

Portal vein Doppler

The normal portal vein (PV) flow is a continuous monophasic flow above baseline with minor variations (see Supplementary material online, *Figure S6*). As venous congestion worsens, the portal flow becomes pulsatile, with decreases in flow during RV systole. In extreme cases, flow can be interrupted or even have a retrograde component giving a to-and-fro appearance. Portal pulsatility can be quantified using the portal pulsatility index (PPI). The portal pulsatility index was defined as (VMax – VMin/VMax) * 100%. Here, VMax is the maximal velocity and VMin is the minimal velocity during the cardiac cycle. We also examined PPI as a continuous variable to assess whether small changes in PV flow was a clinically important marker of venous congestion.

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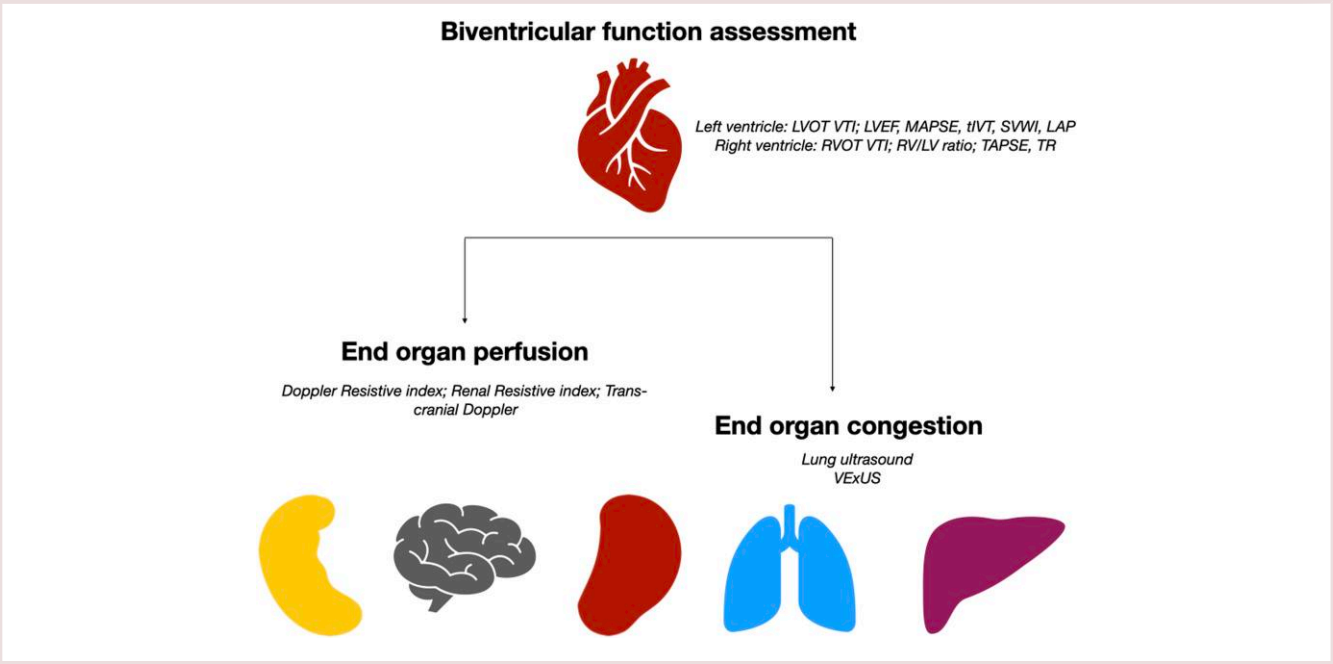
Biventricular function assessment
Left ventricle: LVOT VTI; LVEF, MAPSE, tIVT, SVWI, LAP
Right ventricle: RVOT VTI; RV/LV ratio; TAPSE, TR
End organ perfusion
Doppler Resistive index; Renal Resistive index; Trans-cranial Doppler
End organ congestion
Lung ultrasound
VExUS

Figure 3 Suggested workflow for the evaluation of the organ perfusion and congestion. LV VTI, left ventricular velocity–time integral; LVEF, left ventricular ejection fraction; MAPSE/TAPSE, mitral/tricuspid annular plane systolic excursion; LVSWI, left ventricular stroke work index; t-IVT, total isovolumic time.

Intra-renal venous Doppler

Intra-renal venous Doppler is normally a continuous monophasic flow below the baseline, which progressively becomes first interrupted with two phases, analogous to the S- and the D-waves of the hepatic vein (HV) flow. Similar to the HV pattern, as venous congestion worsens, the S-wave becomes smaller and the D-wave is more pronounced. Eventually, the S-wave disappears entirely, leaving only a monophasic D-wave. The intra-renal vein (IRV) Doppler waveforms were considered abnormal if either a biphasic or monophasic renal vein flow pattern was present. This was defined as discontinuous venous flow with either a systolic/diastolic pattern or a diastolic only pattern.

Quantitative models for congestion

Venous excess ultrasound

In an effort to create a comprehensive assessment of the splanchnic venous system's degree of congestion, an effort was made to derive a scoring system encompassing the IVC and the above-described Doppler morphologies of the portal, hepatic, and intra-renal veins.[61] The resultant venous excess ultrasound (VExUS) score, when elevated, was found to have a strong association with splanchnic organ failure as measured by renal dysfunction, and the finding has been consistent in subsequent studies.[62,63] The advantage of using several vantage points on the splanchnic venous circulation is that it avoids the potential false positives or negatives that can occur with any single measurement or Doppler pattern.

The assessment begins with the finding of an enlarged IVC (>2 cm), which represents the junction and physiological relationship of the MSFP and the RAP, keeping the IVC pitfalls in mind, and is the first step suggesting the possible occurrence of splanchnic venous congestion.[57] In order to confirm and eventually quantify the severity, the HVs, PVs, and IRVs are interrogated, and the score rises with worsening Doppler envelope morphology abnormalities (*Figure 2*).

In the shock patient, the VExUS score can be used in a number of potentially useful ways. Firstly, in the initial resuscitation phase, it can be used as a fluid stop point. A VExUS score suggestive of congestion (1–2) physiologically suggests that further fluid may be detrimental to organ function, and a score of 3 suggests that there may already be a level of congestion that impacts organ function. Secondly, in the de-resuscitation phase, it can again guide the clinician towards more aggressive fluid removal with a higher VExUS score, particularly in the patients with acute kidney injury, where a superimposed congestive insult may be even more significant.

Workflow

While we have outlined a thorough system-based diagnostic strategy to phenotype and quantify shock, we do not intend for this protocol to be followed in its totality and in this precise order in all patients. Rather, clinicians should view this as an assembly of tools which can be used to assist in diagnosis and therapeutic management. That being said, we recommend that all patients in shock receive an assessment of cardiac function (both RV and LV function) and a global assessment for peripheral perfusion including both the adequacy of arterial supply and the presence and degree of venous congestion.[31] Further studies can be conducted as per the specific patient's clinical presentation. *Figure 3* outlines our recommended ultrasonographic workflow.

Conclusion

Point-of-care ultrasound is an essential tool for any clinician that manages patients with shock. The assessment of both perfusion and congestion leads treatment teams to directed therapies. Additional work is required to clarify which ultrasonographic techniques and clinical protocols are most informative for individual patient populations.

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Author contributions

Guido Tavazzi (PhD) (conceptual design, writing, figure creation, reviewing, references), Rory Spiegel (writing, reviewing figure creation), Philippe Rola (writing, reviewing figure creation), Susanna Price (conceptual design, writing, reviewing), Francesco Corradi (conceptual design, writing, reviewing), and Maxwell Hockstein (writing, reviewing figure creation)

Supplementary material

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Data availability

This manuscript does not include patients data.

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